



# UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE  
United States Patent and Trademark Office  
Address: COMMISSIONER OF PATENTS AND TRADEMARKS  
Washington, D.C. 20231  
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/919,770	07/31/2001	Paul Bornstein	UOFW117618	4001

26389 7590 01/31/2003

CHRISTENSEN, O'CONNOR, JOHNSON, KINDNESS, PLLC  
1420 FIFTH AVENUE  
SUITE 2800  
SEATTLE, WA 98101-2347

EXAMINER

GIBBS, TERRA C

ART UNIT PAPER NUMBER

1635

DATE MAILED: 01/31/2003

9

Please find below and/or attached an Office communication concerning this application or proceeding.

**Office Action Summary**

Application No.

09/919,770

Applicant(s)

BORNSTEIN ET AL.

Examiner

Terra C. Gibbs

Art Unit

1635

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☐ Responsive to communication(s) filed on \_\_\_\_.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 1-7 and 10-18 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-7 and 10-18 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☒ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on \_\_\_\_ is: a) ☐ approved b) ☐ disapproved by the Examiner.
- If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

**Priority under 35 U.S.C. §§ 119 and 120**

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
- a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

**Attachment(s)**

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 5.
- 4) ☐ Interview Summary (PTO-413) Paper No(s). \_\_\_\_.
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: \_\_\_\_\_.

### **DETAILED ACTION**

This Office Action is a response to the Election filed 11/4/02, in Paper No. 8.

Claims 1-7 and 10-18 are pending.

Claims 8, 9 and 19-27 have been canceled. Applicant timely made an election without traverse to the restriction requirement filed on 11/4/02, in Paper No. 8.

Claims 1-7 and 10-18 have been examined as they read on the elected subject matter.

#### ***Election/Restrictions***

Applicant's election without traverse of Group I (claims 1-7 and 10-18) filed on 11/4/02, in Paper No. 8 is acknowledged.

#### ***Specification***

The specification is objected to because the specification at page 7, lines 4 and 18, recites the terminology "<http://www.ncbi.nlm.nih.gov/blast>". Embedded hyperlinks and/or other forms of browser-executable code are impermissible and must be deleted. The attempt to incorporate subject matter into the patent application by reference to a hyperlink and/or other forms of browser-executable code is considered to be an improper incorporation by reference. See MPEP 608.01(p), paragraph I regarding incorporation by reference. Furthermore, if the application should issue and be placed on the Office web page, the URL may be interpreted as a valid HTML code and become a live web link, transferring an user to a commercial web site. Office policy does not permit the Office to link to any commercial site because the Office exercises no

Art Unit: 1635

control over the organization, views or accuracy of the information contained on these outside sites.

***Claim Rejections - 35 USC § 112***

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-7 and 10-18 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The instant claims read on a thrombospondin 2 antagonist, wherein the thrombospondin 2 antagonist is a thrombospondin 2 antisense nucleic acid.

The scope of the claimed invention encompasses compounds that target a broad genus of thrombospondin 2 genes that are not properly described in the specification as filed. The claimed invention encompasses nucleic acid compounds that specifically hybridize all forms of the thrombospondin 2 gene, which includes sequences from other species, mutated sequences, polymorphic and allelic variants, splice variants, sequences that have an unspecified degree of identity (similarity, homology), and so forth. The specification as filed provides only a description of the thrombospondin 2 (see SEQ ID NO. 3).

The specification provides a description of the thrombospondin 2 gene (SEQ ID NO. 3). However, the specification as filed, does not provide a sufficient description that would allow

Art Unit: 1635

one of skill in the art to use SEQ ID NO. 3 to predict the structures of *any/all* antisense thrombospondin 2 nucleic acids, including these isolated from other sources, including all polymorphic, allelic and splice variants of this gene.

See the Guidelines for Examination of Patent Applications Under the 35 USC 112 ¶ 1, “Written Description” Requirement (Vol. 66, No. 4, pages 1099-1111). These guidelines state that: “To satisfy the written description requirement, a patent specification must describe the claimed invention in sufficient detail that one skilled in the art can reasonably conclude that the inventor had possession of the claimed invention. An applicant shows possession of the claimed invention by describing the claimed invention with all of its limitations using such descriptive means as words, structures, figures, diagrams, and formulas that fully set forth the claimed invention. Possession may be shown in a variety of ways including description of an actual reduction to practice, or by showing that the invention was “ready for patenting” such as by the disclosure of drawings or structural chemical formulas that show that the invention was complete, or by describing distinguishing identifying characteristics sufficient to show that applicant was in possession of the claimed invention.”

Additionally, “[T]he skilled artisan cannot envision the detailed chemical structure of the encompassed polynucleotides and/or proteins, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. The nucleic acid itself is required. See Fiers v. Revel, 25 USPQ2d 1601, 1606 (CAFC 1993) and Amgen Inc. V. Chugai Pharmaceutical Co. Ltd., 18 USPQ2d 1016. In Fiddes v. Baird, 30 USPQ2d 1481, 1483, claims

Art Unit: 1635

directed to mammalian FGF's were found unpatentable due to lack of written description for the broad class. The specification provided only the bovine sequence.

Applicant's specification does not provide a sufficient number of representative species of antisense thrombospondin 2 nucleic acids which would allow one of skill in the art to predict the structures of all members of the claimed genus of compounds. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Therefore, the specification does not describe the claimed compounds in such full and concise terms so as to indicate that the applicant had possession of these compounds at the time of filing of this application. Applicant is reminded that Vas-Cath makes clear that the written description provision of 35 USC 112 is severable from its enablement provision. (See page 1115.).

Claims 1-7 and 10-18 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Claims 1-7 and 10-18 are drawn to a method for modulating the amount or biological activity of thrombospondin 2 in an animal using a thrombospondin 2 antagonist; wherein the thrombospondin 2 antagonist is a thrombospondin 2 antisense nucleic acid and the thrombospondin 2 antagonist improves the wound response.

The specification at page 8, lines 11-12 contemplates therapeutic applicability of the instant invention to any animal, including mammals, such as human beings. The specification at

Art Unit: 1635

page 23, in Example 1, discloses that wild type mice implanted with a device including a surface collagen layer including an antisense thrombospondin 2 construct displayed an increase in foreign body capsule blood vessel density.

The specification does not provide particular guidance or particular direction for a method for modulating the amount or biological activity of thrombospondin 2 using a thrombospondin 2 antagonist; wherein the thrombospondin 2 antagonist is a thrombospondin 2 antisense nucleic acid and the thrombospondin 2 antagonist improves the wound response in a human being. The specification does not provide guidance for the delivery of an antisense nucleic acid into the target organ and target cells in a human being in quantity sufficient to modulate the amount or biological activity of thrombospondin 2. The specification provides no particular nexus between the increase in foreign body capsule blood vessel density in mice (see Example 1 or Figure 6) to modulating the amount or biological activity of thrombospondin 2 to improve the wound response in humans leaving one in the art to perform undue trial and error experimentation to practice the invention over the scope claimed. The specification provides no particular guidance or direction for addressing the problems of targeting, permanence and quantity of expression of the gene in question, immunogenicity, etc, for nucleic acid/antisense targeting of thrombospondin 2 in humans. The specification provides no nexus between the increase in foreign body capsule blood vessel density in mice, and the modulation of the amount or biological activity of thrombospondin 2 in a human being, as contemplated by the specification. The specification provides no particular guidance or direction for the improvement of the wound response in a human being using a thrombospondin 2 antisense nucleic acid of the claimed invention.

Kyriakides et al. (Journal of Controlled Release, 2002 Vol. 78:295-303) disclose that the efficacy of antisense treatments to decrease thrombospondin 2 expression is difficult to assess. Kyriakides et al. further disclose, “the variability of thrombospondin 2 expression prevented us from accurately determining the extent of reduction in thrombospondin 2 expression”.

Kyriakides et al specifically address the unpredictability associated with the antisense therapeutic art with regard to thrombospondin 2. Furthermore, the art shows that the antisense art is unpredictable in general, which demonstrates further the unpredictability of the claimed invention. For example, Crooke (Stanley T. Crooke, 1998, Basic Principles of Antisense Therapeutics, Springer-Verlag, NY, pages 1-50), states “extrapolations *from in vitro* uptake studies to predictions about *in vivo* pharmacokinetic behavior are entirely inappropriate and, in fact, there are now several lines of evidence in animals and man [that] demonstrate that, even after careful consideration of all *in vitro* uptake data, one cannot predict *in vivo* pharmacokinetics of the compounds based on *in vitro* studies” (see Crooke, page 3).

Branch (TIBS, February 1998 Vol. 23, pages 45-50) further addresses the level of unpredictability of the art of antisense therapy with the following statements: “Antisense molecules and ribozymes capture the imagination with their promise of rational drug design and exquisite specificity. However, they are far more difficult to produce than was originally anticipated, and their ability to eliminate the function of a single gene has never been proven.”; “To minimize unwanted non-antisense effects, investigators are searching for antisense compounds and ribozymes whose targets sites are particularly vulnerable to attack. This is a challenging quest.”; “However, their unpredictability confounds research application of nucleic acid reagents.”; “Non-antisense effects are not the only impediments to rational antisense drug



Art Unit: 1635

design. The internal structures of target RNAs and their associations with cellular proteins create physical barriers, which render most potential binding sites inaccessible to antisense molecules.”; “Years of investigation can be required to figure out what an ‘antisense’ molecule is actually doing,...”; “Because knowledge of their underlying mechanism is typically acting, non-antisense effects muddy the waters.”; “Because biologically active compounds generally have a variety of effects, dose-response curves are always needed to establish a compounds primary pharmacological identity. Antisense compounds are no exception. As is true of all pharmaceuticals, the value of a potential antisense drug can only be judged after its intended clinical use is known, and quantitative information about its dose-response curve of conventional drugs, which typically span two to three orders of magnitude, those of antisense drugs, extend only across a narrow concentration range.”; “Because it is very difficult to predict what portions of an RNA molecule will be accessible *in vivo*, effective antisense molecules must be determined empirically by screening large number of candidates for their ability to act inside cells.”; “Binding is the rare exception rather than the rule, and antisense molecules are excluded from most complementary sites. Since accessibility cannot be predicted, rational design of antisense molecules is not possible.”; and, “The relationship between accessibility to oligonucleotide (ODN) binding and vulnerability to ODN-mediated antisense inhibition *in vivo* is beginning to be explored...It is not yet clear whether *in vitro* screening techniques...will identify ODN’s that are effective *in vivo*.”

Jen et al. (Stem Cells, 2000, Vol. 18:307-319) discuss antisense-based therapy and the challenges that remain before the use of antisense becomes routine in a therapeutic setting. Jen et al. discuss the advances made in the art but also indicate that more progress needs to be made

Art Unit: 1635

in the art. In the conclusion of their review, Jen et al. assert, "Given the state of the art, it is perhaps not surprising that effective and efficient clinical translation of the antisense strategy has remained elusive." It is also stated, "The key challenges to this field have been outlined above. It is clear that they will have to be solved if this approach to specific antitumor therapy is to become a useful treatment approach. A large number of diverse and talented groups are working on this problem, and we can all hope that their efforts will help lead to establishment of this promising form of therapy." It is clear from Jen et al. that the state of the art of antisense is unpredictable and those highly skilled in the art are working towards making the art of antisense therapy more predictable but have many obstacles to overcome.

One of skill in the art would have to engage in trial and error experimentation to develop a method for modulating the amount or biological activity of thrombospondin 2 using a thrombospondin 2 antagonist; wherein the thrombospondin 2 antagonist is a thrombospondin 2 antisense nucleic acid and the thrombospondin 2 antagonist improves the wound response in a human being, that employ the thrombospondin 2 antisense oligonucleotides of the claimed invention. In view of the unpredictability of the art, the quantity of experimentation required would include the de novo determination of how to engineer and deliver an antisense nucleic acid to modulate the amount or biological activity of thrombospondin 2 such that a wound response in a human being would be improved to any degree. Particularly, in view of the obstacles needed to overcome to use of antisense nucleic acids as therapeutics as exemplified in the references discussed above.

Art Unit: 1635

Claims 1-7 and 10-18 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Claims 1-7 and 10-18 are drawn to a method for modulating the amount or biological activity of osteopontin in an animal using a molecule consisting of osteopontin; wherein the molecule consisting of osteopontin improves the wound response. The specification at page 11, lines 18-27 discloses that amount and/or biological activity of osteopontin in an animal can be modulated by the delivery of nucleic acid molecules encoding osteopontin into the body of an animal, for example.

The specification at page 8, lines 11-12 contemplates therapeutic applicability of the instant invention to any animal, including mammals, such as human beings. The specification at page 27, in Example 2, discloses that osteopontin null mice demonstrate high levels of foreign body giant cells surrounding an implant. The specification at pages 28-30, in Example 3, discloses that osteopontin immobilized in the surface layer of an implanted device causes a reduction of both fibrous capsule thickness and macrophage infiltration surrounding an implanted device.

The specification does not provide particular guidance or particular direction for a method for modulating the amount or biological activity of osteopontin using a molecule consisting of osteopontin; wherein the molecule consisting of osteopontin improves the wound response in a human being. The specification does not provide guidance for the delivery of a molecule consisting of osteopontin into the target organ and target cells in a human being in

Art Unit: 1635

quantity sufficient to modulate the amount or biological activity of osteopontin. The specification provides no particular nexus between the reduction of fibrous capsule thickness and macrophage infiltration surrounding an implanted device (see Example 3 or Figure 8A) to modulating the amount or biological activity of osteopontin to improve the wound response in humans leaving one in the art to perform undue trial and error experimentation to practice the invention over the scope claimed. The specification provides no particular guidance or direction for addressing the problems of targeting, permanence and quantity of expression of the gene in question, immunogenicity, etc, for gene therapy targeting osteopontin in humans. The specification provides no nexus between the reduction of fibrous capsule thickness and macrophage infiltration surrounding an implanted device in mice, and the modulation of the amount or biological activity of osteopontin in a human being, as contemplated by the specification. The specification provides no particular guidance or direction for the improvement of the wound response in a human being using a molecule consisting of osteopontin of the claimed invention.

O'Regan et al. (International Journal of Experimental Pathology, 2000 Vol. 81:373-390) assert that osteopontin is a multifunctional protein, however the precise role of osteopontin *in vivo* remains unclear (see page 378, first column).

See the statements above of Crooke, Branch and Jen et al. regarding the level of predictability or unpredictability associated with *in vivo* nucleic acid delivery and therapy.

One of skill in the art would have to engage in trial and error experimentation to develop a method for modulating the amount or biological activity of osteopontin using a molecule consisting of osteopontin; wherein the a molecule consisting of osteopontin improves the wound

Art Unit: 1635

response in a human being, that employ the molecule consisting of osteopontin of the claimed invention. In view of the unpredictability of the art, the quantity of experimentation required would include the de novo determination of how to engineer and deliver a molecule of osteopontin to modulate the amount or biological activity of osteopontin such that a wound response in a human being would be improved to any degree. Particularly, in view of the obstacles needed to overcome to use of nucleic acids as therapeutics as exemplified in the references discussed above.

### ***Claim Rejections - 35 USC § 102***

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in-

(1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effect under this subsection of a national application published under section 122(b) only if the international application designating the United States was published under Article 21(2)(a) of such treaty in the English language; or

(2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that a patent shall not be deemed filed in the United States for the purposes of this subsection based on the filing of an international application filed under the treaty defined in section 351(a).

Claims 1 and 11 are rejected under 35 U.S.C. 102(b) as being anticipated by Liaw et al.

(Journal of Clinical Investigation, 1998 Vol. 101:1468-1478).

Claims 1 and 11 are drawn to a method of modulating the amount or biological activity of osteopontin in an animal using a molecule of osteopontin; wherein osteopontin is introduced into the animal.

Liaw et al. disclose the generation of osteopontin null mice (see Abstract). Liaw et al. further disclose osteopontin null mice were generated via injection of an transgene osteopontin vector in C57BL/6 mice (see page 1469, first column). Liaw et al. further disclose osteopontin protein expression was not detected in kidney tubules from osteopontin mutant animals (see Figure 2D) and osteopontin transcript was absent in homozygous mutant mouse embryonic fibroblast and kidney cells (see Figure 2C).

Claims 1 and 11 are rejected under 35 U.S.C. 102(b) as being anticipated by Gardner et al. (Oncogene, 1994 Vol. 9:2321-2326).

Gardner et al. disclose the reduction in osteopontin synthesis by antisense RNA expression in transformed fibroblast cells (see Abstract and Figure 1B). Gardner et al. further disclose an osteopontin antisense construct expressed in B77 Rat1 cells was injected into nude mice and formed less lung tumors than the cells expressing the osteopontin sense construct (see Figure 1B).

Claims 1 and 11 are rejected under 35 U.S.C. 102(a) as being anticipated by Okada et al. (American Journal of Physiological and Renal Physiology, 2000 Vol. 278:F110-F121).

Okada et al. disclose antisense oligodeoxyribonucleotides targeting osteopontin was intravenously administered to Goodpasture syndrome rats (see Abstract). Okada et al. further

Art Unit: 1635

disclose osteopontin mRNA and protein levels decreased in whole nephritic kidney 12 hours after antisense injection (see Figures 6 A and B).

Claims 1-7 and 10 are rejected under 35 U.S.C. 102(e) as being anticipated by Streit et al. [U.S. Publication No: 2002/0119921].

Claims 1-7 and 10 are drawn to a method for modulating the amount or biological activity of thrombospondin 2 in an animal using a thrombospondin 2 antagonist; wherein the thrombospondin 2 antagonist is a thrombospondin antisense nucleic acid or ribozyme; wherein the thrombospondin 2 antagonist is introduced into the animal; wherein the amount or biological activity of thrombospondin 2 is decreased; wherein the thrombospondin 2 antisense nucleic acid is at least ninety percent identical to the complement of SEQ ID NO:3 and wherein the thrombospondin 2 antisense nucleic acid hybridizes under stringent conditions to SEQ ID NO:3.

Streit et al. disclose a method of modulating thrombospondin 2 (TSP-2) activity by administering a TSP-2 antisense or TSP-2 ribozyme that binds to cellular TSP-2 mRNA and inhibits (decreases) expression of the protein (see page 5 [0049]). Streit et al. further disclose the level of TSP-2 activity is decreased by intravenously administering a TSP-2 antisense or TSP-2 ribozyme (see page 5 [0050]). Streit et al. further disclose the method of modulating thrombospondin 2 (TSP-2) activity by administering a TSP-2 antisense or TSP-2 ribozyme that binds to cellular TSP-2 mRNA and inhibits (decreases) expression of the protein is performed *in vivo* (see page 5 [0051]). Streit et al. further disclose the nucleotide sequence of TSP-2 (see Streit et al. SEQ ID NO. 1) and the amino acid sequence of TSP-2 (see Streit et al. SEQ ID NO. 2). The nucleotide and amino acid sequences of Streit et al. are identical to SEQ ID NOs. 3 and

Art Unit: 1635

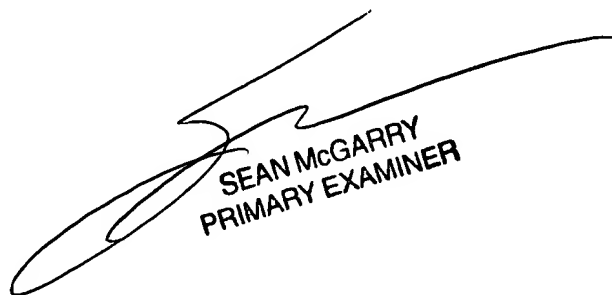
4 of the instant invention, respectively. Note also MPEP 2112.01 that states, "if the prior art teaches the identical chemical structure, the properties applicant discloses and/or claims are necessarily present."

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Terra C. Gibbs whose telephone number is (703) 306-3221. The examiner can normally be reached on M-F 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, John L. LeGuyader can be reached on (703) 308-0447. The fax phone numbers for the organization where this application or proceeding is assigned are (703) 746-8693 for regular communications and (703) 872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is (703) 308-0196.

tcg  
January 23, 2003



SEAN MCGARRY  
PRIMARY EXAMINER